RECURRENCE OF GASTRIC ULCER AFTER COMPLETE VAGOTOMY*

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DIVISION OF THE VAGUS NERVE supply to the stomach has been performed on 521 patients at The University of Chicago Clinics during the six year period from January, 1943, to January, 1949. In only 20 patients was the operation done for the treatment of gastric ulcer. Because the chief problem in gastric ulcer is the possibility of cancer, subtotal resection was performed whenever the lesion would permit its removal with a sufficient margin of normal tissue to have some significance as a therapeutic measure, should subsequent microscopic study show the presence of carcinoma. Total gastrectomy was not considered wise in the absence of proved cancer. Vagotomy was thus a substitute for a partial gastrectomy which would either not remove the lesion at all or be inadequate in the presence of carcinoma. A number refused gastrectomy altogether but were willing to have the less mutilating vagotomy. A report of the first eight patients was made two years ago[1] and the problem is now reconsidered because subsequent experience has produced some modifications in our views. It is now clear that vagotomy is not as effective in gastric ulcers as we have found it to be in duodenal and gastrojejunal ulcers. We have not yet encountered a duodenal ulcer that has failed to heal or has recurred when the vagotomy has been complete as determined by repeated physiological tests. Failures have been observed, but these have all occurred after incomplete vagotomy in patients where a positive gastric secretory response to insulin hypoglycemia has been obtained on repeated testing, and the nocturnal hypersecretion has not been reduced.

Of the group of 20 patients with gastric ulcers, 17 had vagotomy alone, two vagotomy plus gastroenterostomy, and one vagotomy plus partial gastrectomy. At the present time 11 of these patients are entirely free of symptoms on an unrestricted diet and without medication. One died at home two months after operation, of a cerebral hemorrhage; autopsy was not secured, so the status of the ulcer is undetermined. One patient died of a brain tumor five months after operation. Autopsy in this case revealed that the gastric ulcer had healed. One patient had a vagotomy plus a wedge resection of the ulcer on the lesser curvature. Gastroenterostomy was subsequently performed because of obstructive symptoms, and the patient died from a transfusion

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reaction due to mis-matched blood. In one patient the diagnosis of a gastric ulcer on the lesser curvature was based on roentgenogram findings. Vagotomy was performed, although at the time the surgeon could not be certain of the presence of an ulcer. Symptoms persisted, together with positive roentgen ray findings, and 19 months later a sub-total gastrectomy was performed. No ulcer was found in the resected specimen, and inspection of the short cardiac segment also failed to disclose an ulcer. Persistence of symptoms after the resection indicates that the original diagnosis was in error, and that the patient never had an ulcer. One patient had a severe hemorrhage from a large gastric ulcer on the lesser curvature six weeks after vagotomy. The ulcer was later excised and the patient is well at the present time. Four patients continued to have ulcer distress 4 months, 5½ months, 13 months and 22 months after vagotomy. The first three of these patients submitted to sub-total gastrectomy, and in each case an unhealed gastric ulcer was found in the resected specimen.

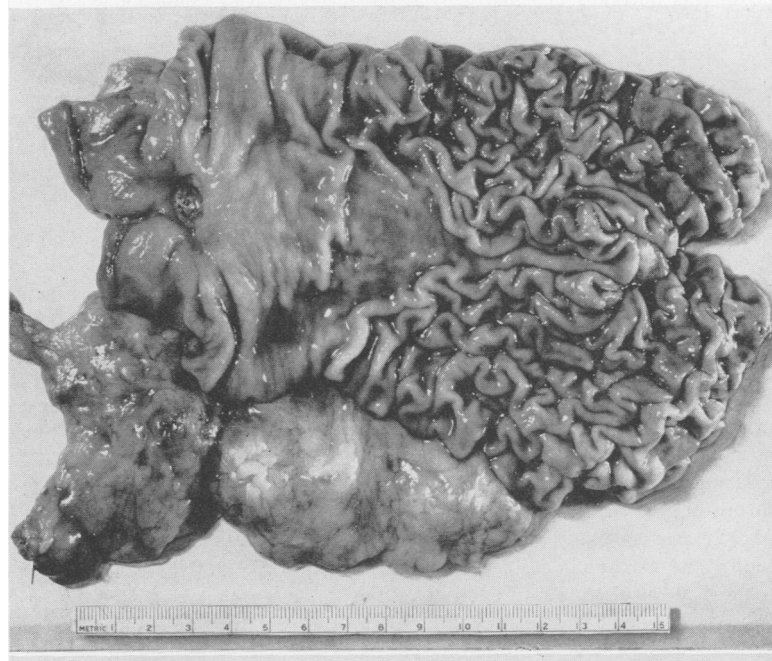
It is thus apparent that in at least five of the 20 patients with gastric ulcers, vagotomy failed to bring about the symptomatic relief and objective evidence of healing usually observed following this operation in duodenal and gastrojejunal lesions. In three of the five patients the vagotomy was complete, as evidenced by persistently negative responses to insulin hypoglycemia, together with a satisfactory reduction in the output of acid from the fasting stomach. Summaries of these cases follow.

A. N., a 66-year-old housewife, was hospitalized in October, 1947, with the complaint of typical abdominal distress for the previous four months. She had had one tarry stool three months previously and had had some episodes of vomiting but no hematemesis. There had been a progressive anorexia, with a 30-pound weight loss. In September, 1947, roentgenograms revealed a very large penetrating ulcer on the lesser curvature of the proximal third of the body of the stomach. This measured approximately 2½ cm. in diameter. Nocturnal secretion measured 415 cc., with a free acid of 36 clinical units. On October 17, 1947, exploratory laparotomy was performed and a large penetrating ulcer adherent to the liver was found on the lesser curvature of the stomach near the cardia. An abdominal vagotomy was performed. She made an uneventful recovery and was discharged on the twelfth postoperative day. She was readmitted on November 30, 1947, because of recurrent hematemeses during the previous two weeks, so that she became progressively weak, and had anorexia and recurrent severe vomiting episodes. On the day of admission she was exsanguinated and in a state of impending shock. Supportive measures were instituted, but the hemorrhage persisted intermittently, and on the ninth of December the abdomen was reopened and the ulcer excised locally. The patient recovered, following a stormy postoperative course, and was discharged from the hospital on January 16, 1948. On January 4, 1948, gastric secretory studies were made, and a night secretion varying from 420 to 750 cc., with 25 to 57 units of free acid, was obtained, this despite the fact that there was a large food residue. The insulin test was also interpreted as being probably positive. Roentgen ray examination in January, 1948, revealed a marked deformity of the mid-portion of the stomach with a narrow channel connecting the two portions, and there was marked gastric retention. The patient's subsequent course has been remarkably uneventful. She has gained 27 pounds to February, 1949, and was eating everything without distress. Further roentgen ray examination of the stomach has not been made.

T. B., a 50-year-old salesman, was admitted on January 23, 1947, complaining of typical ulcer distress of three years' duration. He had vomited frequently, and for the past month the pain had been very severe, especially at night. Two years previously he had an episode of tarry stools. Roentgen ray examination in December, 1946, showed a large ulcer crater in a deformed duodenal bulb without obstruction. Roentgenogram of the gallbladder revealed stones. On January 25, 1947, his night secretion was 2440 cc. with a free acidity of 39 clinical units and a total acid output of 95 milliequivalents. On January 27, 1947, vagotomy and cholecystectomy were performed under spinal anesthesia. The ulcer was found to be on the gastric side of the pylorus. Three weeks after operation the patient had recurrence of ulcer distress accompanied by vomiting. Roentgen ray examination on February 19, 1947, showed persistence of the crater in the pyloric canal, but it was smaller in size. Distress persisted, and on March 11, 1947, roentgen ray examination showed that the crater was still present. On March 27, 1947, the night secretion was 840 cc. with no free acid, and on May 13, 1947, 650 cc. with no free acid. Two insulin tests were negative. On May 16, 1947, a sub-total gastrectomy was performed. Examination revealed the resected ulcer to be located just on the gastric side of the pylorus on the lesser curvature (Fig. 1). Microscopically it was benign. Eight months after operation he was relieved of ulcer distress but could not eat fatty foods and had lost about eight pounds in weight. When last seen in December, 1948, he had no ulcer pain, but complained of moderate diarrhea.

W. H. R., a 32-year-old farmer, first came to the clinic on May 7, 1948. He gave a history of epigastric distress and pain under the lower sternum beginning in November, 1941, and persisting with temporary intermissions to the time of admission. The pain was usually constant, but was relieved by the ingestion of food and alkalies. One episode of severe hematemesis with associated tarry stools occurred in 1941, requiring repeated transfusions. Roentgen ray examination revealed an ulcer in the stomach. There were no further episodes of bleeding, but the epigastric distress persisted in spite of a fairly accurate medical management. Fluoroscopic examination on May 8, 1948, failed to reveal a lesion in the stomach or duodenum. The night secretion averaged 550 cc. with no free acid. A laparotomy was performed on May 10, 1948, and the abdomen was carefully explored without finding any evidence of disease. The stomach and duodenum appeared normal throughout, and there were no scars or adhesions present. The pylorus was patent. The stomach was not opened, but in view of the previous episodes of hemorrhage with symptomatology of peptic ulcer, a vagotomy was performed. Convalescence was complicated by more than the usual amount of gastric stasis, although there was complete relief of the former epigastric distress. Fluoroscopic examination on August 2, 1948, showed marked gastric stasis but there was no evidence of an organic lesion in the stomach or duodenum. The nocturnal gastric secretion on October 31, 1948, averaged 330 cc. in a 12-hour period, with no free acid and 27 clinical units of total acidity. The insulin test was negative. Recurrence of epigastric distress similar to that experienced before the vagotomy was noted in October, 1948, and this rapidly became more severe. Gastroscopic examination on October 30, 1948, revealed an ulcer interpreted as benign on the anterior wall of the mid-portion of the body of the stomach. Fluoroscopic examination on November 2, 1948, revealed a gastric ulcer with a crater, demonstrated by a niche 25 mm. in length, along the midportion of the lesser curvature of the stomach. A second gastroscopic examination on November 1, 1948, again revealed a large benign ulcer on the anterior wall near the lesser curvature in the mid-portion of the stomach. On November 3, 1948, a sub-total gastric resection was performed. The resected specimen revealed a large, typical, benign gastric ulcer on the posterior wall of the stomach near the lesser curvature (Fig. 2). The ulcer crater measured 1.5 cm. in length by 7 mm. in width and 5 mm. deep. The wall surrounding the ulcer was thickened and the mucosa granular. Microscopically there was marked infiltration of the mucosa with round cells, including plasma cells, and

FIG. 1



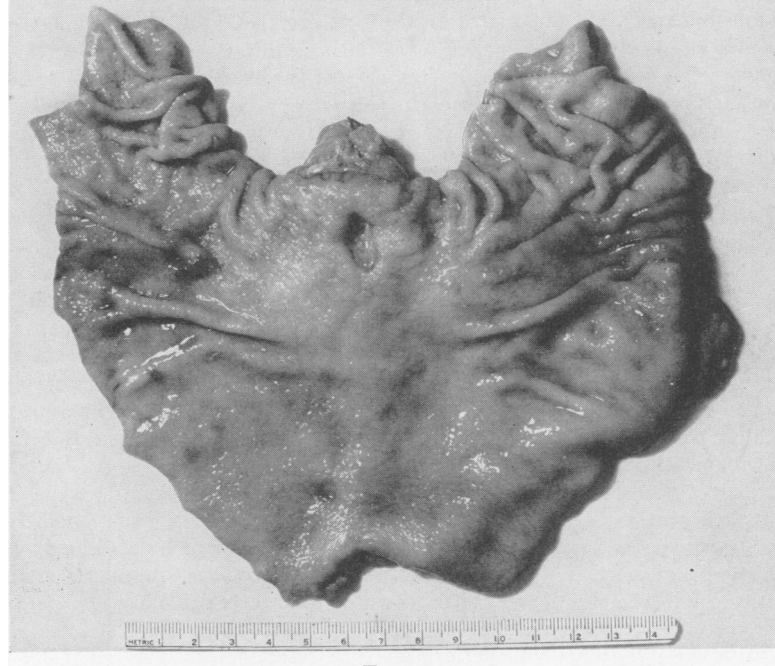


FIG. 2

FIG. 1.—Photograph of resected stomach showing benign pre-pyloric ulcer four months after vagotomy with negative insulin tests afterward.

FIG. 2.—Photograph of resected stomach showing benign gastric ulcer five and one-half months after complete vagotomy.

near the muscularis mucosa there were numerous large reactive lymphoid nodules. The sub-mucosa was fibrotic and invaded by lymphocytes, plasma cells, and eosinophils. The serosa, however, and the mesogastrium, were thickened and hyperemic, and the fatty tissue somewhat fibrotic. The ulcer had penetrated the main muscle coats and undercut the gastric mucosa, particularly on its distal border. Its surface was made up of exudate. Immediately under this region was quite dense granulation tissue with focal fibrinoid degeneration. Recovery from the partial gastrectomy was uneventful, and when last seen on April 4, 1949, the patient was feeling quite well, working every day, and was without epigastric distress or diarrhea. He complained of moderate feelings of fullness and distention after eating.

P. B., a 64-year-old physician, gave a history of intermittent gastric pain and distress for 14 years, with roentgenogram demonstrating a duodenal ulcer on several occasions. There had been one episode of hematemesis in 1933; and he had had recent night pain. The patient brought outside roentgenograms which demonstrated a healed duodenal ulcer and a new ulcer crater on the lesser curvature of the stomach; according to the history this crater had originally been seen in 1939. There was no history of obstruction or co-existent disease. Medical management had been good. Laparotomy on November 24, 1947, showed a scar and an adhesion of the old ulcer in the first portion of the duodenum. There was no evidence of obstruction. On the lesser curvature of the stomach on the mid-section was an indurated area with a perforating ulcer whose crater could be felt. A subdiaphragmatic vagotomy was performed. Postoperatively there was a temperature elevation of 38 to 39 degrees rectally for two days which may have been due to a mild atelectasis. A six-week follow-up showed moderate diarrhea, decreasing in severity since the time of operation. There was no ulcer distress but there were mild symptoms of stasis. The patient's weight gain progressed to 26 pounds in November, 1948, when, for the first time, ulcer distress recurred. An insulin test was negative seven months postoperatively but was positive on December 14, 1948; nocturnal secretion was of low volume with no free acidity. Roentgen ray revealed a lesser curvature gastric ulcer and a sub-total gastrectomy was performed on December 15, 1948. A benign ulcer, recurrent at a previously involved site, was found on pathologic examination of the specimen. In January, 1949, the patient was making a favorable convalescence without ulcer distress.

H. A., a 46-year-old machinist, was admitted to the clinic complaining of typical ulcer distress for the previous eight years. He had had no night pain, no hemorrhages, no perforation, and no obstructive symptoms. Medical management had been excellent and had included a course of roentgen ray therapy to the fundus of the stomach. Repeated roentgen ray examinations since 1942 had demonstrated a recurring, benign, gastric ulcer with hour-glass deformity of the stomach. On November 22, 1946, the night secretion was 861 cc., with no free acid, and the insulin test was positive. On December 4, 1946, a transabdominal vagotomy was performed under spinal anesthesia. A firm, freely movable mass could be felt high on the lesser curvature of the stomach. On December 12, 1946, one insulin test was negative. Gastroscopy on July 2, 1947, showed considerable gastric retention and a possible superficial ulcer. In October, 1947, he complained of recurrent epigastric distress which was considered to be ulcer distress. Roentgen ray examination on November 5, 1947, revealed an hour-glass contraction but no definite crater. Eighteen months postoperatively he continued to have pains similar to his preoperative distress. Roentgenogram on June 23, 1948, revealed persistent deformity of the greater curvature of the stomach with no evidence of a crater.

The development of benign gastric ulcer in patients who have previously had a vagotomy for duodenal ulcer with subsequent healing presents an interesting phenomenon. We have had one proved case of this type and there is possibly a second as yet unconfirmed.

The first patient to have a complete vagotomy in this clinic, W. B., a white male, age 51 years, had been operated upon in 1930 for a perforated peptic ulcer. He felt well until 1938, when he developed epigastric pain which was most severe two hours after the noon meal and at 2:00 A.M. This pain was relieved by food, alkalies, rest, or vomiting. Fluoroscopic examination on December 8, 1942, revealed high-grade pyloric stenosis with a large ulcer crater. Nocturnal gastric secretion averaged 1160 cc. with a free acidity of 65 clinical units, and a total hydrochloric acid output of 75.4 milliequivalents. On January 18, 1943, a transthoracic vagotomy with resection of 3 cm. of both vagus nerves was performed. The ulcer distress promptly disappeared and the nocturnal gastric secretion was reduced to 310 cc. with a free acidity of 58 clinical units and a hydrochloric acid output of 18 milliequivalents. There was evidence of marked stasis of food in the stomach, but the patient continued free from distress and in good general condition for about four years. In 1947, typical ulcer distress recurred and gastroscopy revealed a large ulcer on the posterior wall of the stomach near the lesser curvature. The insulin test was repeatedly

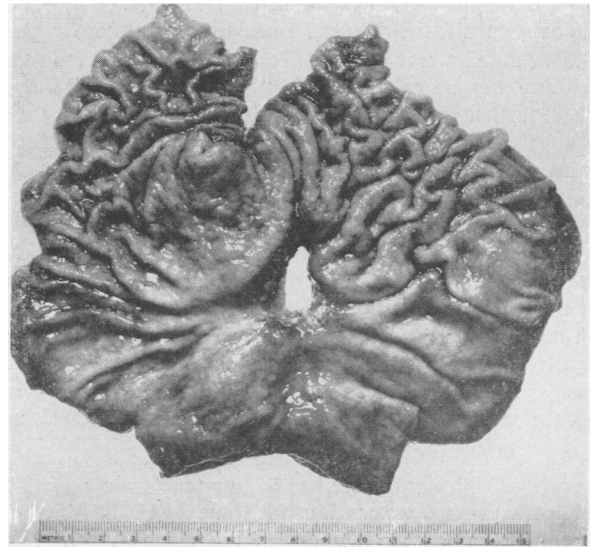
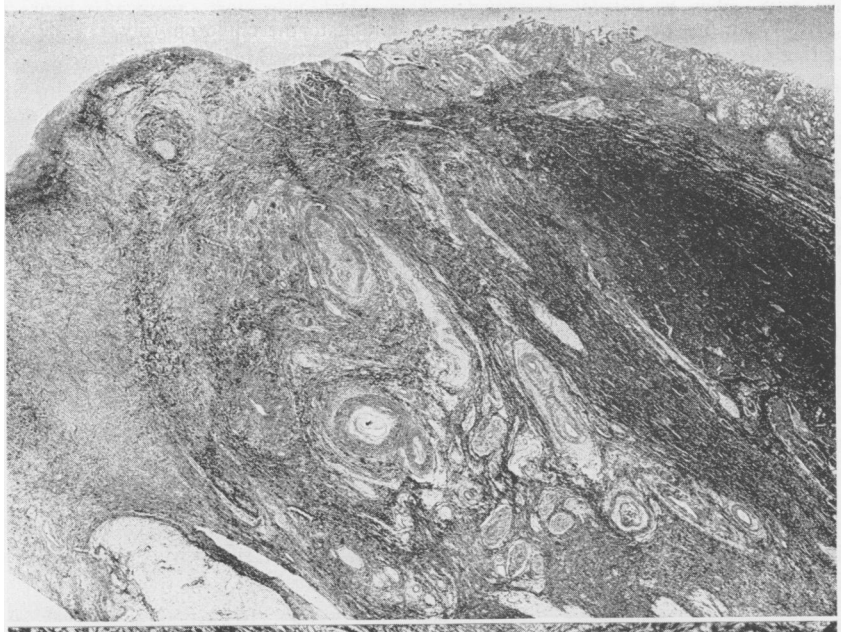


FIG. 3.—Photograph of resected stomach showing gastric ulcer penetrating into the pancreas which developed four years after complete vagotomy for duodenal ulcer. Duodenal ulcer remained healed.

negative and the nocturnal gastric secretion averaged 620 cc. with no free acid. A carcinoma was suspected and a sub-total gastrectomy was performed April 11, 1947. The resected specimen disclosed a large benign gastric ulcer (Fig. 3) with marked intimal proliferation and thrombosis in the blood vessels in the neighboring gastric wall (Figs. 4 and 5).

Of a total of nine patients in our series with both gastric and duodenal ulcers five had vagotomy alone and four vagotomy plus gastroenterostomy. One patient has been lost to follow-up and six are free of symptoms at the present time. One died of intra-cranial hemorrhage six weeks after the vagotomy and at autopsy the duodenal ulcer was healed, the gastric ulcer partially healed. One patient died in the immediate postoperative period of

FIG. 4



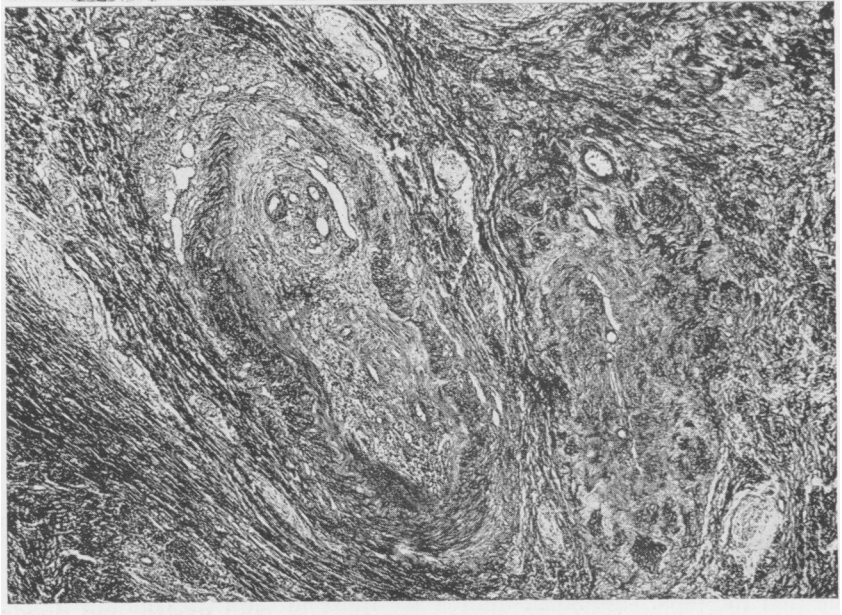


FIG. 5

FIG. 4.—Photomicrograph showing intimal proliferation, thrombosis, and re-canalization in vessels in gastric wall near the ulcer seen in Figure 3.

FIG. 5.—Higher magnification showing details of thrombosis, organization, and re-canalization of vessels in Figure 4.

persistent bleeding from the gastric ulcer. This history is summarized as follows:

R. W., a 65-year-old white male, was admitted to the clinic on July 11, 1947, complaining of typical ulcer distress for the previous 30 years. Medical management had been inadequate. He was in profound alkalosis as a result of persistent vomiting when he came to the hospital. Fluoroscopic examination revealed high-grade pyloric obstruction with marked gastric retention. On July 20, 1947, following hematemesis and melena, the patient went into vascular collapse necessitating repeated transfusions. On July 22, 1947, he was transferred to surgery, where supportive therapy was continued, and the patient received 3 more transfusions. Although still in a precarious state and with moderate alkalosis, on July 24, 1947, a laparotomy was performed. Exploration of the stomach revealed a crater on the lesser curvature, 3 cm. from the esophagus, and a duodenal ulcer with obstruction. A vagotomy and posterior gastroenterostomy were performed. The postoperative course was stormy with persistent high fever, and the blood pressure frequently fell to shock levels, and the patient required oxygen. On July 29, 1947, he had a massive internal hemorrhage, and five blood transfusions were given. The blood pressure stabilized on the following day, but then he had another massive hemorrhage, and accordingly, on July 30, 1947, he was re-explored. The stomach was opened and found to be filled with a large blood clot which, when removed, revealed a large shallow ulcer crater on the lesser curvature. In the center of the ulcer an arteriosclerotic artery was seen spurting blood. This was ligated with a mattress suture and the abdomen closed. The patient's condition failed to improve in spite of repeated transfusions and other supportive measures. Aspiration pneumonia developed, and death occurred on August 13, 1947.

TABLE I.—*Average Twelve-Hour Night Gastric Secretion in the Empty Stomach of Normal Individuals and Peptic Ulcer Patients.*

	Number of Cases	Volume in CC.	Free Acid in Clinical Units	HCl Output in Milli-equivalents
Normals	33	581	31	18
Normals (prison)	23	621	44	30
Duodenal ulcers before vagotomy	135	1,085	52	60
Duodenal ulcers after vagotomy	70	521	22	11
Gastric and duodenal ulcers before vagotomy	8	1,106	38	42
Gastric and duodenal ulcers after vagotomy	8	459	12	5.5
Gastric ulcers before vagotomy	14	773	23	17.8
Gastric ulcers after vagotomy	10	465	3	1.4

In our previous report[1] we commented on the fact that hypersecretion of gastric juice was not found in patients with gastric ulcer, although this was the usual finding in duodenal ulcer. Subsequent experience has confirmed this observation, and is well illustrated in Table I, which displays the volume, free acidity, and total acid output in the nocturnal secretion of patients with various types of peptic ulcer compared with normal individuals. It is probable that the most significant figures are those in the fifth column, recording the total output of hydrochloric acid in the empty stomach in a 12-hour period. This figure is obtained by multiplying the volume of secretion in liters by the concentration of hydrochloric acid in clinical units. The product represents the 12-hour output of hydrochloric acid in milliequivalents. The volume and free acid concentration are of lesser meaning, since there is usually

contamination of the parietal secretion by saliva or regurgitated duodenal content.

Thirty-three normal individuals secreted an average of 18 milliequivalents of hydrochloric acid, and 14 gastric ulcer patients 17.8, or almost an identical amount. In contrast to this, 135 patients with duodenal ulcers secreted 60 milliequivalents, or three times the normal value. Eight patients with both gastric and duodenal ulcers secreted 42 milliequivalents of hydrochloric acid. In the duodenal ulcer patients the nocturnal acid secretion was reduced to 11 milliequivalents by vagotomy, or less than the normal level, and in the gastric ulcers the secretion of acid was almost abolished. In the patients with both gastric and duodenal ulcers there was also a marked reduction in acid secretion to 5.5 milliequivalents.

Several recent authors[2, 3] have called attention to striking differences in gastric and duodenal ulcers with respect to total incidence, sex incidence, response to therapy, and other factors. Surgeons have been impressed with the fact that whereas gastrojejunal ulcers frequently were seen following gastroenterostomy or small gastric resections for duodenal ulcer, they were almost never seen when similar operations were done for gastric ulcer. Differences have now been demonstrated in gastric secretion and in the response of these lesions to complete vagotomy. Can these observations be harmonized with the experimental studies on the pathogenesis of ulcer?

The capacity of pure gastric juice to erode the normal mucosa of the stomach and intestines and thus produce a progressive peptic ulcer has been amply demonstrated by controlled experiments in laboratory animals.[4] Ulcers do not appear under normal conditions because the gastric content is made up of not only gastric juice but substances such as food, saliva, pyloric mucus, and duodenal secretions which dilute and buffer the pepsin-hydrochloric acid. These neutralizing factors constitute a local protective mechanism against ulcer formation which is usually adequate. Removal of the duodenal secretions from the upper intestinal tract reduces the efficiency of this protective mechanism, and when this is done in experimental animals as in the method of Mann and Williamson,[5] ulcers regularly are produced. A defect of this type has been demonstrated to occur only rarely in man[6] and probably plays a small role in the clinical problem of ulcer disease. A decreased production of gastric mucus, which comes chiefly from the antrum, has been sought for in ulcer patients, and while some suggestive findings have been reported, no definite conclusions can be made. On the other hand, an increase in the corrosive properties of the gastric content due to hypersecretion of gastric juice in response to the physiologic stimulus of food, and more importantly in the empty stomach as well, has been demonstrated in the great majority of duodenal ulcer patients. An excessive nocturnal secretion in these cases has been almost invariably present in our experience.[7, 8] Both the hypersecretion of the empty stomach and the exaggerated response to food intake have been abolished by complete removal of the vagus innervation of the stomach,

indicating that they are of neurogenic origin. Usually, relief of ulcer distress and objective evidence of healing have paralleled the decrease in acid in the gastric content, and both may be ascribed to this effect.

In gastric ulcer, however, a hypersecretion of gastric juice has not been found, and as a consequence the corrosive properties of the gastric content due to the concentration of hydrochloric acid and pepsin are not greater than in normal individuals. This would suggest that these ulcers are due to a local loss of resistance to gastric digestion, which may in some cases be vascular thrombosis, as called for in the theory of Virchow and Hanser and exemplified in case W. B. in this report.

We cannot conclude that digestion of the gastric wall by pepsin-hydrochloric acid plays no role in the genesis of gastric ulcers, since as W. L. Palmer[9] has repeatedly pointed out, acid is always present in these cases if carefully looked for, and medical management with antacid therapy cures many of them. Also it should be pointed out that most of the gastric ulcers in our series healed following vagotomy, and that this healing was usually accompanied by a marked reduction or abolition of free acid in the night secretion.

The concept of a primary difference in pathogenesis between gastric and duodenal ulcers may be useful in directing therapy. Hypersecretion with resultant increase in the corrosive properties of the gastric content in duodenal ulcers calls for measures to reduce the hypersecretion. Since this hypersecretion is neurogenic in origin, vagotomy appears to be the logical procedure. Vagotomy is clearly less indicated in gastric ulcers, since hypersecretion is not present, and in any case the possibility of cancer dictates a partial gastrectomy whenever practicable.

Gastrojejunal ulcer clearly belongs with duodenal ulcer in pathogenesis. Here a previously normal area of jejunal mucosa succumbs when exposed to the digestant action of the gastric content. It is not digested away by the normal content, as is evidenced by the absence of stoma ulcers when gastroenterostomy is done for gastric ulcers or pyloric cancers, or when performed in normal dogs. Stoma ulcers are commonly found following gastroenterostomy in duodenal ulcer patients with excessive nocturnal secretion. The fact that antrum resection does not prevent gastrojejunal ulcers is further evidence that nervous factors are more important in these lesions, and this conclusion is borne out by the favorable response of these ulcers to vagotomy.

A deleterious effect of gastric stasis is indicated in this study. The patient who developed a gastric ulcer four years after vagotomy for duodenal ulcer had persistent severe retention. Although he had a stenosing duodenal ulcer, a supplementary gastroenterostomy was not performed because the vagotomy was done by the transthoracic approach and at that stage in our study it was considered essential to determine the effect of the vagotomy alone. Two of the patients with gastric ulcers that failed to heal following vagotomy had persistent gastric stasis. A supplementary gastroenterostomy was not usually

done in patients with gastric ulcers because this was thought to be unwise in the absence of organic obstruction.

While the major cause of gastric stasis following vagotomy is undoubtedly the marked reduction in the tonus and motility of the body of the stomach following the procedure, an important factor in many cases is pylorospasm of varying degree. This is indicated by the fact that when gastroenterostomy was performed in addition to vagotomy in 262 patients in our series, retention in the immediate postoperative period was less and resumption of normal emptying occurred sooner than in those with vagotomy alone. Since we are now persuaded that vagotomy protects against the development of gastrojejunal ulcer, posterior gastroenterostomy has been adopted as a routine procedure when vagotomy is performed for duodenal ulcer. It is quite possible that had gastroenterostomy been done in addition to vagotomy in the gastric ulcers in this series, the results might have been better.

In a recent report[10] Finsterer gives an account of 614 operations he has performed on ulcers in the region of the cardia. In 79 of these, the ulcer was left in situ and the lower half of the stomach removed after the method of Kelling and Madlener. The operative mortality was 5 per cent, and about 90 per cent of the patients remained free of pain. The beneficial effect of this operation probably depends on the reduction in gastric secretion secured through removal of the antrum. In a series of experimental studies, my associates E. R. Woodward and R. R. Bigelow, and I have recently determined that of the total gastric juice produced in dogs, roughly 40 per cent is secreted in response to vagal stimuli, 40 per cent from the stimulation from the antrum, and 20 per cent from the intestines. It is accordingly probable that vagotomy produces just as great a reduction in gastric secretion in gastric ulcer patients as would be secured by the Kelling-Madlener operation, and if combined with a gastroenterostomy to control the factor of pylorospasm, would accomplish just as much by a more conservative operation.

CONCLUSIONS

1. In a group of 17 patients with gastric ulcers treated by vagotomy alone, the ulcer failed to heal or recurred in five or 29 per cent. In three of these the vagotomy was complete.

2. In a group of 197 patients with duodenal ulcers treated by vagotomy alone, gastric ulcers subsequently developed in two, although the vagotomy was complete and the nocturnal hypersecretion abolished in both instances.

3. In a group of 262 patients with duodenal ulcers treated by vagotomy and gastroenterostomy, no gastric ulcers have so far appeared.

4. In patients with duodenal ulcers, and in those with co-existing duodenal and gastric ulcers, the output of hydrochloric acid in the nocturnal fasting secretion is from three to four times the amount in normal individuals.

5. In patients with gastric ulcers the output of hydrochloric acid in the nocturnal fasting secretion is the same or less than that in normal people.

6. Complete vagotomy produces a much greater decrease in the output of hydrochloric acid in duodenal than in gastric ulcers (a ratio of 49 to 16.4 milliequivalents).

7. The absence of gastrojejunal ulcers following partial gastrectomy or gastroenterostomy for gastric ulcer or pyloric cancer in man or following gastroenterostomy in dogs indicates that the jejunal mucosa can resist the digestant action of the gastric content when the secretion of gastric juice is within the normal range, or depressed.

8. The frequent occurrence of stoma ulcers following gastroenterostomy or antrum resection for duodenal ulcers indicates that the jejunal mucosa cannot resist the digestant action of the gastric content when hypersecretion exists, and furthermore, that this hypersecretion is not abolished by removal of the antrum.

9. The healing of duodenal ulcers following vagotomy and the absence of gastrojejunal ulcers following combined vagotomy and gastroenterostomy, indicates that both of these ulcers are due to the increased corrosive properties of the gastric content as a result of hypersecretion.

10. The absence of hypersecretion in gastric ulcers suggests that these lesions are not due to an increase in the corrosive properties of the gastric content but rather to a decrease in resistance on the part of the gastric wall.

11. Vagotomy is not indicated in the treatment of gastric ulcers because hypersecretion of neurogenic origin is not present, and because sub-total gastrectomy may accomplish something in the way of therapy should the lesion prove to be cancerous.

12. For juxta-esophageal ulcers vagotomy and gastroenterostomy will probably accomplish as much as the Kelling-Madlener operation. Total gastrectomy is not warranted in these cases in the absence of proved cancer. Sub-total gastrectomy does not remove these lesions with a sufficient margin to have therapeutic significance should the disease prove cancerous.

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